In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 15-1137V
(to be published)

PITEY MORGAN, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Chief Special Master Corcoran Filed: December 4, 2019 Influenza Vaccine; Transverse Myelitis; Neuromyelitis Optica Spectrum Disorder; Chronic Demyelination; Evidence Supporting Diagnosis

Sylvia Chin-Caplan, Law Office of Sylvia Chin-Caplan, LLC, Boston, MA, for Petitioner.

Amy P. Kokot, U.S. Dep't of Justice, Washington, D.C., for Respondent.

ENTITLEMENT DECISION[1]

Pitey Morgan filed a petition on October 7, 2015, seeking compensation under the National Vaccine Injury Compensation Program ("Vaccine Program").[2] Petition ("Pet.") at 1 (ECF No. 1). Mr. Morgan alleged that he developed longitudinally extensive transverse myelitis ("LETM") due to the influenza ("flu") vaccine he received on October 16, 2012. *Id.*

An entitlement hearing was held in this matter on January 23, 2019. After consideration of

[1] This Decision will be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its current form. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-10–37 (2012) (hereinafter "Vaccine Act" or "the Act"). Individual section references hereafter shall refer to § 300aa of the Act.

the record and testimony provided at hearing, I find that Petitioner is not entitled to a compensation award. As discussed in more detail below, Petitioner has not offered preponderant evidence to support the alleged diagnosis of LETM, whereas the record evidence preponderates in favor of an alternative diagnosis: Neuromyelitis Optica Spectrum Disorder ("NMOSD"). He also has not established a reliable theory explaining how the flu vaccine could have caused his NMOSD.

I. Factual Background

A. *Medical History Prior to Vaccination*

Prior to receiving the flu vaccine in October 2012, Mr. Morgan had several preexisting health conditions, including lower back pain, lower extremity radiculopathy, multi-level degenerative disc disease, lumbar spondylosis, and prostatitis. The nature and basis for these diagnoses, along with Petitioner's subsequent disease course, has some bearing on the claims asserted herein.

The medical record establishes that Mr. Morgan's lower back pain dated back to August 26, 2009 (three years before vaccination), when he saw Deborah Stayman, PA-C ("PA-C Stayman") for worsening lower back pain with onset three weeks prior. Ex. 4 at 6. He also complained of pain radiating to his left thigh. *Id.* During a physical examination, PA-C Stayman noted that Mr. Morgan exhibited decreased reflexes in his left achilles tendon. *Id.* Suspecting a herniated or bulging lumbar disc, PA-C Stayman ordered an MRI[3] study, which was conducted on September 1, 2009. Ex. 2 at 1; Ex. 4 at 32–33. The MRI results showed "mild foraminal narrowing at the L3–L4 and L4–L5 levels…with moderate foraminal narrowing bilaterally at L5–S1 level. No significant spinal canal narrowing. There are disc bulges involving the lower two lumbar levels." Ex. 2 at 1; Ex. 4 at 32–33.

On September 28, 2009, Mr. Morgan was seen by Anthony Wilson, M.D. Ex. 2 at 9–10; Ex. 4 at 10–11. After reviewing the results of the September MRI, Dr. Wilson referred him to physical therapy. Ex. 2 at 9; Ex. 4 at 10. He later returned to Dr. Wilson on November 2, 2009, and complained of persistent pain that the prescribed physical therapy was not assisting. Ex. 2 at 9; Ex. 4 at 13. Dr. Wilson advised Mr. Morgan to temporarily discontinue physical therapy and

[3] Magnetic Resonance Imaging (MRI) is a diagnostic scanning tool that places the patient in a magnetic field rather than exposing him to radiofrequency signals in a traditional x-ray. *Mosby's Manual of Diagnostic and Laboratory Tests* 1106–07 (5th ed. 2014) (hereinafter "*Mosby's*"). An MRI provides several benefits over CT scans, such as providing better contrast between normal and pathologic tissue as well as not being obscured by bone artifacts. *Id.* at 1107.

ordered an EMG[4] and nerve conduction study[5]. Ex. 2 at 9; Ex. 4 at 13. Mr. Morgan underwent this testing on November 24, 2009, but the results of both tests were found to be within normal limits. Ex. 2 at 12–15; Ex. 4 at 29. He was thereafter given a spinal nerve injection. Ex. 2 at 12; Ex. 4 at 29. He received several more spinal nerve injections between 2009 and 2010. Ex. 2 at 11, 16–17; Ex. 4 at 24.

On January 13, 2011, Mr. Morgan presented to Shoreline Family Medicine and complained of muscle stiffness, decreased range of motion, weakness, and radiating lower back pain. Ex. 5 at 44. During this visit, he was diagnosed with chronic lower back pain and degenerative disc disease, and his Neurontin dosage was increased. *Id.* at 45. He continued to seek treatment at Shoreline Family Medicine on a monthly basis. During these visits, he consistently complained of persistent pain, stiffness, weakness, and radiating lower back pain, though not every symptom was present at every visit. *See id.* at 38–43.

On May 16, 2011, Mr. Morgan returned to Shoreline Family Medicine and reported of dizziness and nausea. Ex. 5 at 36–37. He was diagnosed with vertigo and was treated with medication. *Id.* at 37. In the following months, he continued to complain of dizziness as well as neck pain. *Id.* at 34–35. Then, on June 30, 2011, Mr. Morgan underwent an MRI of his cervical spine, the results of which showed "[s]pondylosis causing some mild to moderate spinal canal stenosis at C5-6 and C6-7. No frank herniated disc is appreciated." *Id.* at 92. These results were reviewed at a follow-up appointment at Shoreline Family Medicine on July 19, 2011, during which Mr. Morgan complained of stiffness, neck pain, lower back pain, and radiating pain. *Id.* at 32–33.

The next year, Mr. Morgan underwent another MRI and x-ray on March 11, 2012, for lower back pain and lower extremity radiculopathy. Ex. 8 at 191, 193. The results of the MRI showed "[m]ulti level degenerative disc disease and lumbar spondylosis with slight interval progression and worsening in the appearance of degenerative change at the L4-5 level." *Id.* at 193. The x-ray performed on Mr. Morgan's lumbar spine demonstrated "no acute disease." *Id.* at 192.

On August 6, 2012, Mr. Morgan returned to Shoreline Family Medicine, complaining of trouble urinating and related concerns. Ex. 5 at 145. Following a physical examination, he was diagnosed with prostatitis. *Id.* at 146. He thereafter returned to Shoreline Family Medicine for a follow-up on September 5, 2012, at which time he complained of stiffness and lower back pain in

[4] An EMG, or electromyography, test is a diagnostic method that measures the response to electrical stimulation of muscle nerves. *Dorland's* Illustrated Medical Dictionary 602 (32 ed. 2012) (hereinafter "*Dorland's*").

[5] Nerve conduction studies are used in conjunction with EMGs to detect and locate peripheral nerve injuries or disease. *Mosby's* at 514.

addition to citing the urological symptoms of frequency and oliguria[6]. *Id.* at 147. During this visit, he was again diagnosed with prostatitis as well as lower back pain and bilateral sciatica. *Id.* at 148.

Mr. Morgan returned to Shoreline Family Medicine on September 24, 2012 and complained of toe and thigh numbness with an onset of three weeks prior, as well as difficulty initiating urination and waking up during the night to urinate. Ex. 5 at 149. A physical examination revealed spinal tenderness and limited range of motion as well as decreased sensation in two of his right toes. *Id.* at 150. Following this visit, Mr. Morgan underwent an ultrasound of his prostate. Ex. 8 at 185. The results of the ultrasound were negative. *Id.*

On October 9, 2012, Mr. Morgan was again seen at Shoreline Family Medicine where he reported lower back and pelvic pain, weakness, poor balance, fatigue, and sleep disturbances. Ex. 5 at 151. He was ordered to undergo a CT scan[7] of his head. *Id.* at 152. The CT scan was performed on October 12, 2012, and the results were unremarkable. Ex. 8 at 166.

B. *Vaccination and Subsequent Concerns for Neurologic Injury*

Mr. Morgan was 54 when he received the seasonal flu vaccination on October 16, 2012. Ex. 1 at 1. The next day (October 17, 2012), Mr. Morgan saw Dr. Arthur Golin for a urologic consultation. Ex. 22 at 7–8. During this visit, Petitioner told Dr. Golin (consistent with the record in this case) that his urinary symptoms had begun the year before, but that "in the last 2 ½ months his situation has deteriorated. It has been progressive and he notes marked hesitance, particularly at night." *Id.* at 8. Mr. Morgan also reported "increasing pain and some weakness in the right lower extremity…numbness, right lateral thigh." *Id.* A physical examination revealed an "enlarged, benign-appearing [prostate] gland" and reduced tone of the anal sphincter. *Id.* at 7. Dr. Golin's assessment was urinary retention—but with a possible neurologic component. *Id.*

On October 22, 2012, Mr. Morgan returned to Shoreline Family Medicine, where he was examined for lower back pain, persistent urinary symptoms, weakness, and radiating pain in his legs. Ex. 5 at 153. He was prescribed medication for his prostatitis and instructed to return in one week for a follow-up appointment. *Id.* at 154. The next day, Mr. Morgan was seen by Scott Greenwald, M.D. at Michigan Pain Consultants for his lower back pain, which Mr. Morgan described as radiating down both of his legs. Ex. 7 at 15. He also reported new numbness in bilateral calves. *Id.* During the visit, he was assessed for bilateral lumbar radiculopathy and lumbar degenerative disc disease, and he was treated with a lumbar epidural steroid injection. *Id.* at 17.

[6] Oliguria is the diminished production and excretion of urine as compared to fluid intake. *Dorland's* at 1318.

[7] A computed tomography (CT) scan employs an emergent x-ray beam measured by a scintillation counter, with the results recorded and processed by a computer for reconstruction display. *Dorland's* at 1935. CT scans are useful when a disease of the central nervous system is implicated and degenerative abnormalities can be identified. *Mosby's* at 1026. A CT scan is generally preferable to an MRI during the initial trauma evaluation and the identification of subarachnoid (hemorrhage) bleeding. *Id.*

A few days later, on October 24, 2012, Mr. Morgan was seen by Justin Grill, D.O. in the emergency room of North Ottawa Community Hospital. Ex. 6 at 20. He was assessed for urinary retention, and again reported that he had been experiencing urinary incontinence issues for about a year. *Id.* During a physical examination, Dr. Grill noted that Mr. Morgan "[m]oves all extremities well." *Id.* at 21. A Foley catheter was placed and Mr. Morgan was instructed to return if he experienced any problems. *Id.* Later that same day, however, Mr. Morgan gradually lost the strength in his legs until he was too weak to ambulate. *Id.* at 16.

Petitioner was thereafter transported by ambulance to the emergency room at Mercy Health where he was evaluated by Christopher Hummel, D.O. for leg weakness and urinary retention. Ex. 6 at 13–19; Ex. 8 at 124. A physical evaluation revealed decreased rectal tone, decreased sensation in his lower legs, saddle paresthesias, and buttocks numbness. Ex. 8 at 125. Following the examination, Dr. Hummel expressed concern for possible cauda equina syndrome[8] and epidural hematoma given Mr. Morgan's history of having a lumbar steroid injection the day prior. *Id.* at 126. An MRI was ordered, and the results showed "[m]ild lumbar disc degeneration, which does not appear significantly changed as compared to 03/11/2012 . . . conus medullaris appears somewhat indistinct with a suggestion of some increased T2-weighted signal intensity, of uncertain significance given the limitations of the low field strength magnet." *Id.* at 140, 143.

Mr. Morgan was subsequently transferred to Mercy Health – Hackley Campus ("Hackley") just a few hours later. Ex. 9 (ECF Nos. 9-1 to 9-3)[9] at 733, 1209. During an initial evaluation conducted by Christopher Marquart, M.D., Petitioner stated that he had first noticed mild weakness in his lower extremities in August 2012 (two months before the vaccination in question) while he was moving his daughter into college. *Id.* at 896. He explained that the patchy numbness and tingling he experienced progressively increased over the previous two months and coincided with his worsening urologic symptoms. *Id.* Dr. Marquart also noted in the history section of the record that Petitioner had recently received the flu shot, and that he reported experiencing occasional blurry vision. *Id.* at 897.

A physical examination revealed that Petitioner had "crude sensory level at about T12-L1 level," as well as "patchy decreased pinprick and light touch over the anterior thighs bilaterally, top of the right foot and bottom both the left heel and patchy over the area of the cath[eter]," decreased strength, and absent reflexes in his lower extremities. Ex. 9 at 898. Dr. Marquart did not note any mass, lesion, or herniated disc in Mr. Morgan's MRI, but he did observe evidence of nerve root clumping and enhancing in the conus—leading him to question whether Mr. Morgan

[8] Cauda equina syndrome is characterized by dull, aching pain of the perineum, bladder, and sacrum that generally radiates in a sciatic fashion. *Dorland's* at 1824. It is typically caused by compression of the spinal nerve roots and is associated with paresthesias. *Id.*

[9] Exhibit 9 was filed as three separate, consecutively-paginated volumes.

had experienced transverse myelitis ("TM") or some other acute, neuro-inflammatory process. *Id.* at 809, 899. Given these concerns, Mr. Morgan was admitted to the intensive care unit for observation, "to make certain he does not have any type of ascending paralysis with the recent flu vaccination." *Id.* at 899.

Mr. Morgan underwent a second MRI on October 25, 2012, the results of which showed "edema within the cord from T8 to the inferior tip of the cord…but no significant contrast enhancement." Ex. 5 at 80. That same day, Mr. Morgan was evaluated by an infectious disease consultant, Roni Devlin, M.D. Ex. 9 at 711. Following a physical evaluation, Dr. Devlin indicated that the MRI was suggestive of myelitis of indeterminate etiology—though he did later implicate the flu vaccine, noting that "[c]ase reports of myelitis following vaccination have certainly been reported, but rarely." *Id.* at 714.

A few days later, on October 27, 2012, Mr. Morgan was evaluated by Larry Wahl, D.O, who noted that Mr. Morgan had experienced "increasing urinary retention and some difficulty with strength in his lower extremities, climbing stairs as much as 5-1/2 weeks ago that gradually increased" and "seemed to reach a critical level 1 day after having an epidural steroid injection on Tuesday [October 23, 2012]." *Id.* at 715. Within his differential diagnosis, Dr. Wahl included viral infection, arachnoiditis, and TM, though he did express some skepticism towards TM as explanatory given the extensive nature of Mr. Morgan's spinal cord edema. *Id.* at 717.

While at Hackley, Mr. Morgan was treated with high dose steroids and intensive physical therapy. *Id.* at 681. He gradually recovered the ability to stand, bear weight, and walk short distances with the assistance of a walker, but he continued to experience numbness and tingling in his lower extremities. *Id.* On October 29, 2012, he was discharged to outpatient rehabilitation and was scheduled to return in one week for a follow-up with Dr. Marquart. *Id.* When Mr. Morgan returned on November 15, 2012, Dr. Marquart reiterated his belief that Petitioner's myelitis was "probably a reaction to his flu vaccine for lack of a better explanation." Ex. 10 at 1. A repeat MRI conducted shortly thereafter on November 21, 2012, showed marked improvement in the appearance of the spinal cord with only "very mild patchy cord edema." Ex. 5 at 79.

On December 13, 2012, Mr. Morgan presented to Douglas Gelb, M.D. at the University of Michigan Neurology Clinic. Ex. 14 at 6. While reviewing Mr. Morgan's history, Dr. Gelb noted that "[h]e has had low back pain for a few years, radiating into one or both legs at times, but never causing numbness or weakness." *Id.* In addition, before the October 16, 2012 vaccination, Mr. Morgan "had noticed some numbness in his right fifth toe for about a month, and would tire a little bit more easily." *Id.* Mr. Morgan also reported that since his hospital discharge on October 29, 2012, he had not noticed much improvement in his ability to ambulate and felt as though his neurologic symptoms were worsening. *Id.* He now specifically complained of persistent loss of sensation in his lower extremities, bladder, and bowls, burning pains, the development of a lump

on his neck, worsening vision, sudden arm jerks, and cramping or spasms in his fingers. *Id.*

Following a physical evaluation—in which Mr. Morgan exhibited mild spasticity in both lower extremities, reduced sensation from the waist down, and absent reflexes in his ankles—Dr. Gelb proposed that Mr. Morgan was experiencing either an isolated episode of TM or the first instance of a recurrent, central nervous system ("CNS") demyelinating disease, such as Multiple Sclerosis[10] ("MS") or Neuromyelitis Optica[11] ("NMO"). *Id.* at 8–9, 11. He acknowledged that Mr. Morgan's pre-vaccination symptoms "raise[d] some concern that he might have had an ongoing disease process in his nervous system that 'flared up' on Oct. 24," but noted that those earlier symptoms were non-specific, or could be explained by Mr. Morgan's degenerative disc disease and enlarged prostate. *Id.* at 10–11. Overall, however, Dr. Gelb did not feel that Mr. Morgan's neurologic disease was progressing, instead attributing his change in vision to dexamethasone—one of the medications Mr. Morgan was taking. *Id.* at 11. He instructed Mr. Morgan to taper off dexamethasone and suggested a follow-up MRI as well as a serum NMO antibodies test. *Id.*

On December 20, 2012, Mr. Morgan returned to Dr. Wahl to discuss his progress. Ex. 11 at 5. During this visit, Mr. Morgan described continuing improvement of his neurologic symptoms, and he demonstrated almost full strength throughout his lower extremities during his physical evaluation. *Id.* In accordance with Dr. Gelb's suggestion, Dr. Wahl ordered laboratory testing—including an NMO serum antibodies test and a brain MRI. *Id.*

C. *Ongoing Treatment of Neurologic Symptoms and Search for Etiology*

Between December 20, 2012 and February 14, 2013, Mr. Morgan exhibited some improvement in his neurologic symptoms, though Dr. Wahl expressed the view that a complete recovery was unlikely, and that any further improvements would be small. *Id.* at 3–5. The result of the NMO serum antibodies test was negative, but, as the results summary remarked, "seronegativity *does not necessarily preclude* a diagnosis of [NMO]." Ex. 5 at 56 (emphasis added). The results of the brain MRI conducted on January 7, 2013 were also negative. *Id.* at 76.

On April 29, 2013, Mr. Morgan returned to Dr. Wahl and reported little improvement but denied any new symptoms. Ex. 11 at 2. On physical examination, he exhibited decreased strength in both legs. *Id.* A repeat thoracic MRI was ordered and performed on May 20, 2013. *Id.*; Ex. 5 at 70. The results showed "[i]nterval change in the appearance of the thoracic spinal cord which

[10] Multiple sclerosis is a disease in which there is demyelination of the central nervous system causing weakness, incoordination, paresthesias, speech disturbances, and visual complaints. *Dorland's* at 1680. Its course is characterized by a series of relapses and remissions. *Id.*

[11] NMO is characterized by the demyelination of the optic nerve and the spinal cord. *Dorland's* at 1266. Symptoms of NMO often include changes in vision, flaccid paralysis of the extremities, and sensory and genitourinary disturbances. *Id.*

demonstrates diffuse but mild expansion and increased intramedullary signal centrally between the T6 level and the conus, the appearance here is suggestive of [TM]." Ex. 5 at 70.

In the weeks following, Mr. Morgan's condition deteriorated such that by June 17, 2013, he was unable to stand independently and exhibited increasingly diminished strength in his bilateral lower extremities. Ex. 11 at 1. He presented to Ivan Landon, M.D. on July 23, 2013 with concerns that he was experiencing a relapse of his symptoms. Ex. 12 at 14. Dr. Landon noted that Mr. Morgan was now paraplegic, whereas he had previously been able to ambulate with the assistance of a walker or cane. *Id.* at 14, 16. During his evaluation, Dr. Landon concluded that Mr. Morgan had suffered at least one, maybe two, relapses and that he was likely suffering from a polyphasic TM. *Id.* at 16. He suggested Mr. Morgan begin inpatient therapy as well as immunoglobulin therapy. *Id.*

On July 31, 2013, Mr. Morgan was admitted to the inpatient rehabilitation unit at Hackley for nine days, during which time he was treated with high dose steroids, IVIG, and intensive physical therapy. Ex. 9 at 2–3. He saw some improvement with these treatments, and was discharged to outpatient rehabilitation on August 8, 2013. *Id.* at 4. Petitioner continued his treatment with Dr. Landon over the ensuing year (between August 2013 and June 2014). Ex. 12 at 1–12. Throughout, Dr. Landon noted that Mr. Morgan's condition appeared to be deteriorating, as he continued to experience recurrent symptoms relapses. *Id.* By June 17, 2014, Mr. Morgan was restricted to a wheelchair and complained of symptoms in his upper extremities. *Id.* at 1. Dr. Landon expressed his frustrations and emphasized that Mr. Morgan seemed to respond best to IVIG coupled with steroids, but that his insurance company was no longer covering the cost of the IVIG treatment. *Id.*

D. *Embrace of NMO Diagnosis in 2014*

On August 15, 2014, Mr. Morgan returned to the University of Michigan Neurology Clinic. Ex. 14 at 93. During this appointment, Mr. Morgan reported that he had developed numbness in his trunk that ascended from his waist to his mid-back, numbness in the tips of his fingers, and blurry spots of vision within the past few months. *Id.* at 93–94. Dr. Gelb conducted a physical examination and found that Mr. Morgan's lower extremities were completely immobile, areflexive, and exhibited reduced sensation to light touch and pain. *Id.* at 95. In his assessment, Dr. Gelb expressed uncertainty as to "whether his clinical deterioration was due to [a] new episode of spinal cord inflammation, or simply some systemic illness exacerbating his deficits from his initial episode (although new episodes of inflammation seem more likely, given the severity and persistence of the new deficits, and given the higher sensory level)." *Id.*

Such concerns prompted Dr. Gelb to refer Mr. Morgan to a MS clinic and order repeat MRIs of Mr. Morgan's cervical and thoracic spine and brain as well as a repeat serum NMO

antibodies test. Ex. 14 at 95–96. The result of Mr. Morgan's serum NMO antibodies test was again negative, but the MRI of his thoracic spine now showed:

> [V]olume retraction/myelomalacia, seen caudal to T8 level and extending down to the conus, is non masslike abnormal enhancement predominantly involving central and posterior portions of the spinal cord, which is more conspicuous at T12 and T10-T11 levels…The spinal cord volume loss likely represent[s] myelomalacia as the sequela of previous inflammatory process. Areas of T2 signal change and abnormal enhancement could represent reactivation of inflammatory process, this possibility should be correlated with deficits on physical exam and paraclinical test/parameters.

Id. at 135, 137. In addition, Mr. Morgan's brain MRI revealed "nonspecific small areas of nonenhancing T2 signal prolongation in predominantly left supratentorial white matter, these findings may represent sequela from previous inflammatory, infectious or small vessel white matter ischemic process." *Id.* at 137.

Following Dr. Gelb's recommendation, Mr. Morgan went to the University of Michigan MS Clinic and was evaluated by Robert Pace, M.D. on November 26, 2014. Ex. 14 at 109. During a physical examination, Mr. Morgan demonstrated reduced tone, absent movement, and absent reflexes in bilateral lower extremities, and absent sensation below midthoracic level. *Id.* at 110. Dr. Pace also reviewed the MRI results from the August 2014 scans, noting:

> [S]everal nonspecific T2/FLAIR hyperintensities seen in the brain. These are not in a pattern that is strongly suggestive of demyelination such as would be seen with [MS]. However, there is T2 hyperintensity in the fourth ventricle surrounding the cerebral aqueduct. This is of unclear significance, but can be seen in [NMO] spectrum….

Id. He also noted "patchy enhancement of the lower thoracic spine/conus medullaris that appears to involve some of the cauda equina." *Id.*

Based on his review of the laboratory testing and imaging studies, Dr. Pace now diagnosed Mr. Morgan with "longitudinal myelitis due to [NMO], sero-negative." *Id.* Dr. Pace emphasized that there was a high likelihood that Mr. Morgan's condition would cause "recurrent and potentially devastating episodes of myelitis if untreated" and therefore advised him to begin immune modulation therapy. *Id.* He remarked that Mr. Morgan had experienced significant improvement with IVIG treatment in the past but opined that the most effective treatment for patients with NMO is Rituximab. *Id.* at 110–11.

Mr. Morgan did not receive either treatment, although he pursued physical therapy from July to September 2015. Ex. 23 at 2; Ex. 8 at 1–11. He then returned to Dr. Pace on August 18, 2015 at the University of Michigan MS Clinic. Ex. 23 at 2. The medical records from the visit list Mr. Morgan's diagnoses as relapsing-remitting MS, Devic's disease[12], and flaccid paralysis of the lower extremities. *Id.* at 1. During the appointment, Mr. Morgan reported persistent paralysis in his lower extremities and numbness from the midthoracic region down. *Id.* at 2. He did, however, feel that he was cognitively doing better than before. *Id.* Dr. Pace ordered repeat MRIs and hepatitis serologies. *Id.* at 3–4. Those MRIs showed that the nonspecific signal hyperintensities located in the periventricular[13] area of the brain were stable since January. Ex. 21 at 1. No cervical spine abnormalities were noted, however, and the previously documented areas of abnormal signal in the thoracic region of the spinal cord had resolved. *Id.* at 5.

Mr. Morgan returned to Dr. Pace on April 20, 2016. Ex. 53 at 40. During this visit, he explained that he had not pursued Rituximab therapy but was seeing improvement with physical therapy. *Id.* at 41. He was still confined to a wheelchair, but he had not developed any new or worsening symptoms. *Id.* Following a physical examination, Dr. Pace again opined that Mr. Morgan's diagnosis was "most likely seronegative [NMO]." *Id.* at 42. Mr. Morgan returned for a follow-up appointment with Dr. Pace the following year, in April 2017. *Id.* at 18. He again reported improvement with continued physical therapy and denied any new or worsening symptoms. *Id.* A physical exam revealed that Mr. Morgan was able to activate his hip flexors and extensors, which were actions he was incapable of performing the year prior. *Id.* at 19. Dr. Pace also reviewed the results of MRIs performed in 2017 and noted that the changes in Mr. Morgan's spine were stable and that there was no evidence of new or enhancing lesions. *Id.* at 19–20. The final diagnoses documented in the differential at the conclusion of the appointment were NMO, acute TM, paralytic syndrome, and spinal stenosis of the cervical region. *Id.* at 21.

II. Witness Testimony

A. *Petitioner's Expert Witness - Dr. Carlo Tornatore, M.D.*

Dr. Tornatore testified at hearing and provided two reports on Petitioner's behalf. *See generally* Ex. 24, filed Oct. 27, 2016 (ECF No. 18-1) ("Tornatore First Rep."); Ex. 47, filed Oct. 5, 2017 (ECF No. 36-1) ("Tornatore Second Rep."). Dr. Tornatore opined that Mr. Morgan developed LETM following his October 16, 2012 vaccination, and that Petitioner's dramatic post-vaccination decline could not be attributed to any of his preexisting conditions. Additionally, he asserted that the close temporal connection between onset and vaccination, plus a lack of alternate explanation, meant that Mr. Morgan's vaccination more likely than not was the reason for his

[12] NMO is sometimes referred to as Devic's disease. *Dorland's* at 503.

[13] The prefix peri- means "near" or "around." *Dorland's* at 1410. Therefore, if a brain lesion is periventricular, it is located near the ventricles of the brain.

condition.

Dr. Tornatore is a board-certified neurologist currently employed in the Department of Neurology of the Georgetown University Medical Center. Curriculum Vitae of Dr. Tornatore, filed Oct. 28, 2016, at 1 (ECF No. 19-1) ("Tornatore CV"). He received his master's and medical degrees from Georgetown University after completing his bachelor's degree from Cornell University. *Id.* at 2. He has been licensed to practice medicine since 1988 and completed an internship in internal medicine at Providence Hospital, a residency in neurology at Georgetown University Hospital, and a fellowship in molecular virology at the National Institute of Health. *Id.* He currently holds an academic position at the Georgetown University Medical Center where he serves as the Residency Program Director as well as the Director of the Neurology Clerkship program for third year medical students. *Id.* at 7. His clinical experience includes serving as the Vice Chairman for the Department of Neurology and Director of the Georgetown University Hospital Multiple Sclerosis Clinic. *Id.* at 3. He has published numerous articles on various neurological issues, and he is frequently invited to lecture on topics within the field of neurology. *Id.* at 7–19.

At hearing, Dr. Tornatore reviewed Mr. Morgan's pre- and post-vaccination medical history in depth. He acknowledged (as several points in the pre-vaccination record reflect) that Mr. Morgan had a significant medical history of lower back pain, radicular symptoms, and bladder issues. Trial Transcript ("Tr.") at 12, 15, 19; Ex. 5 at 146, 148. He emphasized, however, his view that Mr. Morgan's preexisting symptoms were likely attributable to his degenerative disc disease, sciatica and prostatitis—none of which were neurologic in etiology. Tr. 17, 19–20, 25–26; Ex. 4 at 7; Ex. 5 at 148.

In making this distinction, Dr. Tornatore relied on the progression of Mr. Morgan's symptoms, and more specifically, the tempo of that progression. Tr. at 11, 58. He opined that Mr. Morgan's pre-vaccination symptoms had "a very slow progression" that was incongruent with the faster tempo typically associated with LETM. *Id.* at 18, 29. He further explained that a slow, gradual progression of NMO or NMOSD is very uncommon, and in fact is an exclusionary characteristic when diagnosing NMOSD. *Id.* at 30–31; *see also* D. Wingerchuk, et al., *International Consensus Diagnostic Criteria for Neuromyelitis Optica Spectrum Disorders*, 85 Neurology 177, 179 (2015), filed as Ex. E, June 23, 2017 (ECF No. 33) ("Wingerchuk"). Therefore, the drawn-out progression of Mr. Morgan's symptoms prior to vaccination suggested that Mr. Morgan was not at that time suffering from LETM or NMO. Tr. at 58.

After vaccination, by contrast, Mr. Morgan clearly experienced a catastrophic and abrupt collapse. *Id.* at 10, 18; Ex. 9 at 711–12. Dr. Tornatore highlighted the difference between the slow and steady progression of Mr. Morgan's lower back pain over several years and his urologic symptoms over the course of months, versus the accelerated and dramatic deterioration he

experienced one week after receiving the flu shot. Tr. at 10, 18. In light of this sharp decline, Dr. Tornatore opined that Mr. Morgan's pre-vaccination conditions were readily distinguishable from those he experienced post-vaccination. Tr. at 70–72.

Dr. Tornatore also maintained that Petitioner's proper diagnosis was LETM rather than NMOSD. Tr. at 9, 59, 68. Dr. Tornatore described TM as a rare clinical syndrome in which an immune-mediated process causes inflammation within the spinal cord, resulting in scarring and neural injury. Tornatore First Rep. at 2–3. This inflammatory process causes varying degrees of weakness, sensory alterations and autonomic dysfunction. *Id.* LETM is specifically characterized by inflammation that extends through three or more segments of the spinal cord. D. Karussis, et al., *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, Autoimmunity Reviews 1, 6 (2013), filed as Ex. 55, Dec. 19, 2018 (ECF No. 50-4).

In describing the relationship between LETM and NMOSD, Dr. Tornatore espoused the opinion that LETM is an umbrella diagnosis that actually encompasses the more discreet diagnosis of NMOSD. Tr. at 9, 66–67. This position, however, was undercut by the medical literature submitted in support of Petitioner's claim, which described NMOSD as "an inflammatory disease of the [CNS], mostly involving the optic nerve and the spinal cord…and frequently *manifest[ing] as* severe bilateral optic neuritis or *severe longitudinally extensive transverse myelitis*." S. Kim, et al., *Differential Diagnosis of Neuromyelitis Optica Spectrum Disorders*, 10 Therapeutic Advances in Neurological Disorders 265, 265 (2017) (emphasis added), filed as Ex. 49, Dec. 19, 2018 (ECF No. 49-2) ("Kim").

Dr. Tornatore further opined that Mr. Morgan did not meet the strict diagnostic criteria for NMO or NMOSD. Tr. at 48–49, 69. Because Mr. Morgan tested negative for the serum aquaporin-4 antibodies ("AQP4-IgG") normally associated with NMO, and had not been diagnosed with a related disorder within the spectrum, he would have to meet additional, more stringent, diagnostic criteria. *See* Wingerchuk at 179. Patients who are seronegative for AQP4-IgG—like Mr. Morgan—must exhibit two or more core clinical characteristics such as optic neuritis, acute myelitis, area postrema syndrome[14], acute brainstem syndrome, symptomatic narcolepsy, or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions, or symptomatic cerebral syndrome with NMOSD-typical brain lesions. *Id.*

Mr. Morgan, Dr. Tornatore opined, had only exhibited one of these core clinical characteristics: acute myelitis. But this was insufficient to support a diagnosis of NMO or NMOSD because there were no other core clinical characteristics observed, nor was there dissemination in space or time of the transverse myelitis. Tr. at 68. He also noted that Mr. Morgan did not fit the

[14] The area postrema is located within the walls of the brain's fourth ventricle. *See* E. Benarroch, *Circumventricular Organs: Receptive and Homeostatic Functions and Clinical Implications*, 77 Neurology 1198, 1198 (2011), filed on as Court Exhibit 1, Dec. 6, 2019 (ECF No. 63) ("Benarroch"). In NMO, Area Postrema syndrome is characterized by "intractable nausea, vomiting, and hiccups in various combinations." *Id.* at 1202.

demographic profile typically associated with an increased risk of developing such an autoimmune disorder. *Id.* at 49. While Mr. Morgan's disease process had proven to be relapsing and remitting, TM was not always monophasic, and thus a person with TM could experience relapses. *Id.* at 50. Accordingly, Dr. Tornatore concluded that the proper diagnosis for Mr. Morgan's condition was a relapsing and remitting LETM. *Id.* at 9, 59, 68.

Besides offering an opinion on diagnosis, Dr. Tornatore posited that Petitioner's October 2012 flu vaccine likely caused him to develop LETM, through the mechanism of molecular mimicry. Tornatore First Rep. at 3–4; Tr. at 59–60. In support, he referenced several pieces of medical literature that discuss the theory of molecular mimicry and its putative relationship to the development of TM. *See* D. Kerr, et al., *Immunopathogenesis of Acute Transverse Myelitis*, Current Opinion in Neurology 339, 342–43 (2002), filed as Ex. 26, Nov. 7, 2016 (ECF No. 20-1) (discussing the possibility of an immunologically mediated pathogenesis of ATM following immunization and the presence of autoantibodies in patients with recurrent ATM); *see also* N. Nakamura, et al., *Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases*, 42 Internal Med. 191, 193–94 (2003), filed as Ex. 34, Nov. 7, 2016 (ECF No. 20-1) (discussing the theory of molecular mimicry as it applied to a patient who developed TM following receipt of the flu vaccine); C. Wu, et al., *Hemorrhagic Longitudinally Extensive Transverse Myelitis*, Case Reports Neurologic Med. 1, 3 (2016), filed as Ex. 48, Dec. 19, 2018 (ECF No. 49-1) (discussing the likelihood of cross-reactivity between infectious agents and the central nervous system).

Dr. Tornatore also supported his theory of molecular mimicry by referring to statements made by one of Mr. Morgan's primary treating physicians, Dr. Gelb. Tr. at 41–42. In particular, he referenced a note in which Dr. Gelb explained numerous causes of myelitis and proposed that Mr. Morgan's condition was most likely associated with "a one-time activation of the immune system…either triggered by something systemic (such as an immunization, infection, surgery, or trauma) or apparently spontaneous." *Id.* at 42; Ex. 14 at 10–11. This statement, Dr. Tornatore opined, was reliable treater support for the causal relationship between Mr. Morgan's flu immunization and his subsequent condition. Tr. at 42. He emphasized that the time that had elapsed between Mr. Morgan's receipt of the flu vaccine and the onset of his LETM-related symptoms—a period of nine days—was an adequate amount of time for the molecular mimicry mechanism to initiate a demyelinating autoimmune disease. *Id.* at 50.

On cross examination, Dr. Tornatore acknowledged that Mr. Morgan might have experienced a prolonged onset of LETM *prior* to vaccination. *Id.* at 57. He also agreed that many of Mr. Morgan's pre-vaccination symptoms were associated with neuropathies. *Id.* at 52–58. He nevertheless maintained that Mr. Morgan's pre-vaccination symptoms were more likely attributable to unrelated, non-neurologic conditions such as prostatitis and/or degenerative disc disease. *Id.* at 12, 16–17, 20, 25–26. In addition, he reiterated his belief that the temporal

progression of Mr. Morgan's symptoms distinguished his pre- and post-vaccination conditions—the slow, gradual, progression of his pre-vaccination symptoms over several years was characterized as markedly different from the post-vaccination deterioration Mr. Morgan experienced over the course of a few hours on October 24, 2012. *Id.* at 11, 58.

C. *Respondent's Expert Witness: Dr. Subramaniam Sriram, M.D.*

Dr. Sriram testified at hearing and provided two reports on behalf of Respondent. *See generally* Ex. A, filed Jun. 23, 2017 (ECF No. 33-1) ("Sriram First Rep."); Ex. F, filed Dec. 22, 2017 (ECF No. 38-1) ("Sriram Second Rep."). Dr. Sriram, a specialist in neuroimmunology, opined that Petitioner did not suffer from a monophasic occurrence of TM, but rather a clinically relapsing form of LETM, or inflammatory myelitis, nevertheless falling within the overall diagnostic category of NMOSD. Tr. at 82–84, 104; Sriram First Rep. at 5. He also opined that the onset of Mr. Morgan's inflammatory myelitis likely pre-dated his vaccination. Tr. at 96; Ex. A at 6.

Dr. Sriram is a board-certified neurologist with a focus in neuroimmunology. *See* Ex. B, filed Jun. 23, 2017 (ECF No. 33-2) ("Sriram CV"). He obtained a Bachelor of Medicine and a Bachelor of Surgery from the University of Madras in Madras India. *Id.* at 1. He then served as an intern and resident at Wayne State University and completed a residency in neurology at Stanford University, where he also served as chief resident and eventually completed a post-doctoral fellowship in neuroimmunology. *Id.* Currently, Dr. Sriram serves director of the Vanderbilt Multiple Sclerosis Clinic. Tr. at 76. He also holds academic positions as a professor of experimental neurology and therapeutics as well as an associate professor in molecular biology and immunology. Sriram CV at 1. Dr. Sriram's clinical practice includes seeing patients two and a half days a week. Tr. at 76. Additionally, he has published numerous articles about demyelinating diseases and the neuroimmunological pathogenesis for those conditions. Sriram CV at 9–20.

Much like Dr. Tornatore, Dr. Sriram discussed Mr. Morgan's pre- and post-vaccination medical records at length during his testimony. First, Dr. Sriram opined that the neurologic and urologic symptoms Mr. Morgan was experiencing in the months *preceding* vaccination were likely the early manifestations of a demyelinating condition. Sriram First Rep. at 6; Tr. at 92–96. To support this contention, Dr. Sriram cited several records in which Petitioner sought medical treatment for difficulty urinating, toe and thigh numbness, numbness and tingling in his legs, weakness, poor balance, and pelvic pain prior receiving the flu vaccination to October 16, 2012. Ex. 5 at 145–51; Ex. 22 at 8. While he did acknowledge that a slow neurological worsening over months to years is very uncommon in NMOSD, Dr. Sriram emphasized that the symptoms Mr. Morgan experienced signified "a continuum of neurological deficits that began sometime around the end of August, beginning of September [2012]" and ultimately culminated in what Dr. Sriram characterized as a clinically relapsing form of LETM within NMOSD. Tr. at 81–84, 127–28, 160.

Dr. Sriram took issue with Dr. Tornatore's interpretation of the post-vaccination record, and in particular what it says about Petitioner's proper diagnosis. He acknowledged that Mr. Morgan was seronegative for the hallmark antibody associated with NMOSD—AQP4-IgG. Tr. at 106. He explained, however, that some individuals with NMOSD *will* present as seronegative[15] but still otherwise meet the diagnostic criteria for NMOSD. Tr. at 87–92, 108, 110–113; Sriram First Rep. at 6; Wingerchuk at 179. For such a patient to be properly diagnosed with NMOSD, Dr. Sriram explained, they would need to exhibit at least two core clinical characteristics—optic neuritis, acute myelitis, area postrema syndrome, acute brainstem syndrome, symptomatic narcolepsy or acute diencephalic syndrome with NMOSD-typical diencephalic MRI lesions, or symptomatic cerebral syndrome with NMOSD-typical brain lesions—with at least one of the core characteristics being optic neuritis, acute myelitis with LETM, or area postrema syndrome, dissemination in space, and fulfillment of additional MRI requirements, as applicable. Sriram First Rep. at 6; Wingerchuk at 179.

Here, Dr. Sriram maintained, Mr. Morgan met the diagnostic criteria for seronegative NMOSD because he exhibited two of those core clinical characteristics—myelitis *plus* an area postrema brain lesion—and also because the initial lesion extending to T8 on the October 25, 2012 MRI was later found to have extended to T6 on the May 20, 2013 MRI, thereby exhibiting dissemination in space. Tr. at 108, 110–11, 113; Ex. 5 at 70, 80. On cross examination, however, Dr. Sriram conceded that none of the MRI reports in the record unquestionably indicated the presence of a postrema area brain lesion—at most, the record revealed hyperintensity in the periventricular region of the brain where the area postrema is located. Tr. at 114–15, 117; Ex. 21 at 1. Additionally, while the medical records indicate that a brain lesion existed, Dr. Sriram acknowledged that Mr. Morgan had not experienced any symptoms typically associated with area postrema syndrome, though he also noted that it is not uncommon for lesions to be "silent" or asymptomatic. Tr. at 111–12.

During cross examination, Dr. Sriram agreed that "'[r]ecurrent isolated episodes of optic neuritis or myelitis do not qualify [for the diagnosis of NMOSD] in the absence of [evidence of] AQP4-IgG given the broad differential diagnosis of these syndromes.'" Tr. at 115–17 (quoting B, Weinshenker, et al., *Neuromyelitis Spectrum Disorders*, 92 Mayo Clinic Proc. 663, 666 (2017), filed as Ex. G, Dec. 19, 2018 (ECF No. 47). He emphasized, however, that TM is generally considered a monophasic disease—especially when it has a post-infectious etiology—whereas

[15] Dr. Sriram proposed several explanations as to why a patient might be seronegative for this otherwise-critical biomarker of NMOSD. Tr. at 91–92. One such explanation is that the test used to detect the presence of AQP4-IgG has low sensitivity. *Id.* A second explanation is that Mr. Morgan was treated with corticosteroids, which would have suppressed Mr. Morgan's immune system. *Id.* at 92. Additionally, it is understood that, although rare, NMOSD attributable to an infectious process can initially manifest as LETM. Kim at 279. Under such circumstances, a patient will be seronegative for AQP4-IgG. *Id.* at 280.

NMOSD is typically considered to be a chronic condition, with sixty to seventy percent of patients with NMOSD experiencing relapse. Tr. at 86, 90. Dr. Sriram also opined that even if Mr. Morgan's presentation was initially considered characteristic of TM, his subsequent relapse in June 2013 warranted reconsideration of the initial TM diagnosis. *Id.* at 90–91. Taking into account the entirety of Mr. Morgan's clinical course, Dr. Sriram opined, the record suggested that the proper diagnosis was actually clinically relapsing LETM within NMOSD. *Id.* at 82.

III. Procedural History

After this case was initiated and following the filing of pertinent medical records, Respondent filed his Rule 4(c) Report on May 6, 2016, contesting Mr. Morgan's entitlement to damages on May 6, 2016. ECF No. 14. I subsequently ordered the parties to file expert reports in support of their respective positions, and they did so as set forth above. I thereafter set the matter for hearing on January 23, 2019 (ECF No. 41). The hearing took place as scheduled and included testimony from the experts identified above. Following the hearing's conclusion, the parties submitted post-hearing briefs on June 17, 2019 (ECF No. 60 and 61). This matter is now ripe for resolution.

IV. Applicable Legal Standards

A. *Claimant's Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 11(c)(1), 13(a)(1)(A), 14(a); *see also Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[16] In this case, Petitioner does not assert a Table claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(a)(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [they] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enters. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d

[16] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. App'x 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health & Human Services*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Human Servs.*, 121 Fed. Cl. 230, 245 (2015), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017).

In discussing the evidentiary standard applicable to the first *Althen* prong, many decisions of the Court of Federal Claims and Federal Circuit have emphasized that petitioners need only establish a causation theory's biological plausibility (and thus need not do so with preponderant proof). *Tarsell v. United States,* 133 Fed. Cl. 782, 792–93 (2017) (special master committed legal error by requiring petitioner to establish first *Althen* prong by preponderance; that standard applied only to second prong and petitioner's overall burden); *Contreras,* 121 Fed. Cl. at 245 ("Plausibility

. . . in many cases *may* be enough to satisfy *Althen* prong one." (emphasis in original)); *see also Andreu*, 569 F.3d at 1375. At the same time, there is contrary authority from the Federal Circuit suggesting that the same preponderance standard used overall in evaluating a claimant's success in a Vaccine Act claim is also applied specifically to the first *Althen* prong. *See, e.g.*, *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010) (affirming special master's determination that expert "had not provided a 'reliable medical or scientific explanation' *sufficient to prove by a preponderance of the evidence a medical theory* linking the [relevant vaccine to relevant injury]."") (emphasis added). Regardless, one thing remains: petitioners always have the ultimate burden of establishing their Vaccine Act claim *overall* with preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell*, 133 Fed. Cl. at 793 (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *see also Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting *Althen*, 418 F.3d at 1280)). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and/or statements of a treating physician's views, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (finding that it is not arbitrary or capricious for special masters to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Dept. of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr.

Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without op.*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.*; *see also Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *see also Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("Given the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"); *Rickett v. Sec'y of Health & Human Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they

are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[I]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms.").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs*., No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Human Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. denied sub. nom. Murphy v. Sullivan*, 506 U.S. 974 (1992) ("It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.") (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1948)).

There are, however, situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Human Servs*., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); *Lowrie*, 2005 WL 6117475, at *19 ("Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent.") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Human Servs*., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Human Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir.

2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)).

> The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). But nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too

great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs*., No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 Fed. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("Assessments as to the reliability of expert testimony often turn on credibility determinations…."); *see also Porter v. Sec'y of Health & Human Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[T]his court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act.").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all of the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *See Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Human Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. Overview of TM, NMOSD, and Relevant Prior Decisions

The experts largely defined the competing diagnoses in this case correctly, but a few additional points are in order. First, it should be noted that acute demyelinating neurologic conditions like TM are understood to occur rapidly, proceed in a monophasic manner, and often resolve without recurrence (even though they can leave lasting sequelae). *See Palattao v. Sec'y of Health & Human Servs.*, No. 13-591V, 2019 WL 989380, at *11 (Fed. Cl. Spec. Mstr. Feb. 4, 2019) (describing TM's course as "abrupt[]" and "monophasic"). By contrast, chronic demyelinating conditions affecting the CNS, like MS, can initially present as if they were TM but will invariably recur. *See, e.g.*, *Hunt v. Sec'y of Health & Human Servs.*, No. 12-232V, 2015 WL 1263356, at *11 (Fed. Cl. Spec. Mstr. Feb. 23, 2015) (noting that an MS diagnosis traditionally

requires "at least two events disseminated in time and space") (internal quotation marks omitted)), *mot. for review den'd*, 123 Fed. Cl. 509 (2015).

NMOSD is understood to be a relapsing and chronic CNS disease, like MS. It is therefore distinguishable from monophasic conditions like TM, even though both involve CNS demyelination. Wingerchuk at 185 (noting that only "5%–10% of contemporary cases [of NMOSD] are described as monophasic" and requiring five years of relapse-free clinical observation in order to confirm a monophasic course). While a chronic CNS demyelinating disease may *begin* with an occurrence that appears discrete, like TM, the later overall course of disease will establish that the patient did not *only* experience a one-time event.

Such distinctions are critical for purposes of evaluating causation in this case. Program petitioners have on many occasions successfully established that acute forms of CNS demyelinating conditions (e.g., TM or acute disseminated encephalomyelitis ("ADEM")) were likely vaccine-caused. *See, e.g.*, *Raymo v. Sec'y of Health & Human Servs.*, No. 11-0654V, 2014 WL 1092274, at *23 (Fed. Cl. Spec. Mstr. Feb. 24, 2014) (TM injury found to be vaccine caused); *Brown v. Sec'y of Health & Human Servs.*, No. 09-426V, 2011 WL 5029865, at *41 (Fed. Cl. Spec. Mstr. Sept. 30, 2011) (flu vaccine caused Petitioner's ADEM injury); *Banks v. Sec'y of Health & Human Servs.*, No. 02-0738V, 2007 WL 2296047, at *25 (Fed. Cl. Spec. Mstr. July 20, 2007) (awarding compensation for ADEM linked to MMR vaccine); *Kuperus v. Sec'y of Health & Human Servs.*, No. 01-0060V, 2003 WL 22912885, at *11 (Fed. Cl. Spec. Mstr. Oct. 23, 2003) (awarding compensation for ADEM linked to the DTaP vaccine).

By contrast, Program claimants have less consistently succeeded in establishing that a vaccine (including the flu vaccine) could cause a person to develop a chronic demyelinating condition, like MS or NMOSD. *See, e.g.*, *Day v. Sec'y of Health & Human Servs.*, No. 12-630, 2015 WL 8028393 (Fed. Cl. Spec. Mstr. Nov. 13, 2015) (awarding entitlement where HPV and Flumist vaccines were shown to have caused petitioner to develop NMOSD); *Calise v. Sec'y of Health & Human Servs.*, No. 08-85V, 2011 WL 1230155 (Fed. Cl. Spec. Mstr. Mar. 14, 2011) (awarding entitlement for flu/NMOSD injury); *but compare Wei-Ti Chen v. Sec'y of Health & Human Servs.*, No. 16-634V, 2019 WL 2121208, at *22 (Fed. Cl. Spec. Mstr. Apr. 19, 2019) (finding that insufficient evidence was provided to support causal connection between the flu vaccine and petitioner's subsequent development of seronegative NMOSD); *Davis v. Sec'y of Health & Human Servs.*, No. 07-451V, 2010 WL 1444056, *aff'd*, 94 Fed. Cl. 53 (upholding special master's decision that the flu vaccine did not cause NMOSD). Although the results of some of these decisions are consistent with Petitioner's favored outcome, the theories offered are not.

In *Calise and Davis*, for example, the theories offered in both cases associating the flu vaccine with NMOSD relied on the concept that the components of the flu vaccine first caused direct injury to the endothelial cells in the body, thereby producing a breach in the blood brain barrier, and resulting in further injury via a subsequent antibody attack on the myelin sheath (absent

any cross-reactivity via molecular mimicry). *See Calise*, 2011 WL 1230155, at *12–21 (finding that petitioner met her burden of proof by providing medical literature and the opinions of a treating physician which supported her proposed mechanism of causation); *but see Davis*, 2010 WL 1444056, at *8–9 (finding that petitioner did not carry her burden in the absence of medical literature and expert opinion to support her proposed theory of causation). Here, by contrast, Petitioner simply proposes that molecular mimicry between antigens in the vaccine and self-structures of the CNS caused harm, with less explanation as to how the process occurred.

Day involved a causation theory centered on molecular mimicry, as here, but featured two different vaccines acting in concert (i.e., the HPV and Flumist vaccines). The presiding special master decided the claim for petitioner in that case based primarily on record evidence supporting the potential of components of the HPV vaccine to cause a cross-reaction spurred on by AQP4-IgG, for which the claimant tested positive. *Day*, 2015 WL 8028393, at *14. In this case, however, it is undisputed that Mr. Morgan is seronegative, and he did not receive the HPV vaccine. *Day* did not otherwise specifically address the role the flu vaccine by itself might have played in contributing to the petitioner's disease course. *Day*, 2015 WL 8028393, at *14.

The decision most on all fours with the present case is *Wei-Ti Chen*, which I decided. The petitioner in that case had a long-standing history of lower back and buttock pain. *Wei-Ti Chen*, 2019 WL 2121208, at *2. She presented to her chiropractor one week prior to receipt of the flu vaccine complaining of inner thigh tingling as well as persistent lower back and buttock pain. *Id.* Following administration of the flu vaccine, the petitioner developed a demyelinating disease that resulted in differential diagnoses of ADEM, MS, and NMOSD. *Id.* at *3–4. Ultimately the medical record preponderated in favor of an NMOSD diagnosis despite the petitioner's atypical presentation—namely her seronegativity. *Id.* at *20. The petitioner, however, was unable to present sufficient evidence to credibly support a finding that the flu vaccine could cause NMOSD through the mechanism of molecular mimicry. *Id.* at *21–22.[17]

II. Petitioner's Pre-vaccination Symptoms Have Not Been Established to be Associated With His Neurologic Injury

The parties' dispute whether Mr. Morgan's back pain and pre-vaccination urologic problems reflected the onset of some greater neurologic injury (whether TM, NMOSD, or otherwise) can be resolved in Petitioner's favor. The record plainly establishes that Petitioner had a long-standing medical history of documented degenerative disc disease and an enlarged prostate. *See* Ex. 4 at 7; Ex. 5 at 148; Ex. 22 at 9. Some of Petitioner's post-vaccination treating physicians repeatedly offered the view that his pre-vaccination symptoms could be attributable to such pre-existing causes, and it was not unreasonable to consider the possibility that they were related, especially since some of Petitioner's pre-vaccination symptoms (like those pertaining to bladder

[17] Though similar to the present matter, *Wei-Ti Chen* is distinguishable in at least one regard—I found the petitioner's pre-vaccination symptoms *were* a part of her overall disease process. *Wei-Ti Chen*, 2019 WL 2121208, at *23–25.

control) can be neurologic.

Nevertheless, the record preponderates against the conclusion that Petitioner's injury, however characterized, predated his receipt of the flu vaccine. *See, e.g.*, Ex. 14 at 10–11. Dr. Tornatore persuasively established that there was a difference between the tempo of Petitioner's long-standing pre-vaccination symptoms and those he experienced thereafter. In addition, Dr. Sriram seems to have conceded the low likelihood that an individual with his preferred diagnosis of NMOSD would experience a slow and progressive series of symptoms over the relevant time period at issue. Tr. at 127. I therefore do not find, based on this record, that Petitioner's neurologic injury likely predated his October 2012 receipt of the flu vaccine.

III. The Medical Record Best Supports an NMOSD Diagnosis

The parties strenuously disagree on the proper diagnosis of Mr. Morgan. Here, the evidence preponderates *against* Petitioner. Consideration of the record as a whole -- from the time of Petitioner's October 2012 vaccination through the 2016 medical records -- establishes persuasive support for a seronegative NMOSD diagnosis (although admittedly the matter cannot be *conclusively* determined).

First, there is no doubt that treater views support that diagnosis (especially those from physicians who saw Petitioner later in time). *See* Ex. 14 at 110; Ex. 53 at 42. While they are not dispositive of the question by themselves, they offer reliable proof, even in the face of contrary assertions by Dr. Tornatore. *See Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326; *see also Snyder*, 88 Fed. Cl. at 746 n.67. Those treaters did not conclude, as Dr. Tornatore urges, that Petitioner experienced a one-time event that resembled TM, or even (as he allowed) that Petitioner's subsequent course reflected a relapsing form of TM. Indeed, NMO was suspected as early as *December 2012* (just two months after onset). Ex. 14 at 10.

Second, the record upon which treaters based the NMOSD diagnosis preponderantly supports Respondent's position. The University of Michigan MS Clinic records set forth a comprehensive history that strongly supports the NMOSD diagnosis. Ex. 14 at 109–11. Its treaters, including Dr. Pace, acknowledged that Mr. Morgan was seronegative and took into account his initial presentation, but placed it in the context of his greater condition. *Id.* Additionally, the relapsing and remitting nature of Mr. Morgan's disease process, plus the existence of a lesion in the area of the brain most commonly associated with NMOSD, lend further credence to Dr. Pace's conclusion that Mr. Morgan's condition was properly diagnosed as NMOSD. *Id.* at 110. Petitioner is correct in pointing out the criteria that apply in the context of a seronegative patient, as well as the difficulty in establishing those criteria, but there was still evidence to fit each criterion—Petitioner initially exhibited acute myelitis with LETM, and demonstrated brain lesions in the area postrema region of the brain. *See* Ex. 5 at 56; Ex. 14 at 110, 135; Ex. 21 at 1; Ex. 53 at 21; *see also* Wingerchuk at 179.

In response, Petitioner reasonably argued that one of these criteria—evidence of area

postrema syndrome—is *not* strongly supported by the record. Ex. E at 3. Indeed, Mr. Morgan did not even receive such a specific diagnosis. There is, however, as Dr. Sriram proposed, evidence of dissemination in space, because later MRI reports from September 2015 show a brain lesion in the periventricular region of the brain (Ex. 14 at 110; Ex. 21 at 1), as well as a lesion extending to T6, where it had previously extended only to T8 before resolving. Ex. 14 at 137; Ex. 5 at 70, 80. In addition, while Petitioner did not experience some of the *symptoms* that would be associated with an area postrema lesion (Tr. at 111), his treating physicians nevertheless noted that the mere *existence* of an area postrema lesion supported a diagnosis of NMOSD by itself. *See* Ex. 14 at 110. This, along with the overall thrust of treater opinion, better supports the NMOSD diagnosis than Petitioner's arguments to the contrary.

Third, and by contrast, the overall record does *not* preponderate in favor of the TM diagnosis proposed by Petitioner. Treaters initially, and rationally, interpreted Petitioner's symptoms and test results (like MRIs) as supportive of LETM. Moreover, some of the therapies utilized (steroids or PT) proved effective, and Mr. Morgan's improving course, coupled with what appeared to be a lack of radiologic evidence of further lesion activity, all suggested that he had experienced a one-time, monophasic event. Ex. 5 at 79; Ex. 9 at 681. But over time, Petitioner began experiencing a progressive course of symptoms that suggested a relapse, and certainly resulted in more severe symptoms that impacted his ambulation. Ex. 14 at 109. Thereafter, other evidence (as extensively referenced above) undermined the initial conclusion about the possible nature of Petitioner's injury. The overall progressive course of Petitioner's symptoms from October 2012 to 2016 is not supportive of the conclusion that he experienced a one-time acute injury and thereafter suffered its sequelae; rather, the record suggests Petitioner's initial symptoms were part of something chronic that took time to unfold.

Dr. Tornatore's interpretation of the overall record, and Petitioner's symptoms within it, was reasonable but ultimately not persuasive. He allowed that at a minimum, Petitioner had experienced what he deemed a "relapsing" form of LETM—thus implicitly acknowledging that the medical record did *not* reflect a single monophasic injury, as normally would be associated with TM. Tr. at 43–44. He also erred somewhat in asserting that this version of TM would subsume NMOSD - when in fact, as literature filed by Petitioner establishes, the *opposite* is the case. Kim at 265. It is simply more likely than not that NMOSD could present as it did for Petitioner—a conclusion that Mr. Morgan's treaters later embraced, after Petitioner had lived with his symptoms for several years.

Admittedly, the overall record in this case makes it difficult to establish *with certainty* Petitioner's correct diagnosis (a task that I am not even called upon to perform, since diagnosing an illness falls well beyond the purview of the special masters in resolving Vaccine Act claims). Petitioner has raised reasonable objections to this diagnosis, such that I could not find that the NMOSD diagnosis is supported by even 75 percent of the record. However, the evidence still *preponderates* in favor of the NMOSD diagnosis (a determination that merely means more than *50 percent* of the record favors that determination).

IV. Petitioner Has Not Satisfied the *Althen* Prongs

This case largely turns on Petitioner's inability to satisfy the first and second *Althen* prongs.[18] With respect to the first, "can cause" prong, I note that Petitioner's causation theory includes elements that are routinely deemed valid in the Vaccine Program. For example, molecular mimicry has repeatedly been embraced in Program cases as a reliable scientific mechanism for explaining the pathophysiology of certain immune-mediated conditions, including many demyelinating disorders. *See, e.g.*, *Tompkins v. Sec'y of Health & Human Servs.*, No. 10-261V, 2013 WL 3498652, at *22 (Fed. Cl. Spec. Mstr. June 21, 2013) ("[t]he molecular mimicry theory is the one most widely accepted for the agents most frequently accepted as causal"), *mot. for review den'd*, 117 Fed. Cl. 713 (2014). Molecular mimicry has been invoked to explain how a vaccine might cause TM. *See, e.g.*, *Hargrove v. Sec'y of Health & Human Servs.*, No.05-0694V, 2009 WL 1220986, at *38 (Fed. Cl. Spec. Mstr. Apr. 14, 2009).

However, I find preponderant evidence supports a *different* injury—NMOSD. And, as noted above, there is a lack of persuasive authority (in the form of prior Vaccine Act decisions) that suggests this kind of chronic, progressive CNS demyelinating injury has credibly been shown to be associated with vaccination. This is particularly the case if the mechanism of molecular mimicry is considered closely.[19] For molecular mimicry to have utility herein as a reliable mechanism, there should be some evidence that the relevant autoantibodies that are known to drive, or are at least associated with, the resulting demyelinating disease are likely produced as a result of the flu vaccine (or the comparable wild virus) - and it is reasonable to require a petitioner to offer some evidence in support of such a contention when evaluating the success of the claimant's prong one showing. *See, e.g.*, *W.C.*, 704 F.3d at 1361. Petitioner could have established this with a variety of circumstantial evidence involving the flu vaccine (or wild flu virus) and its association with NMOSD, or proof that immune system stimulation can at least *initiate* a chronic process that would indirectly result in the down-stream, continued production of the relevant autoantibodies.

Petitioner, however, offered little such evidence. At best, there are some references in the

[18] Mr. Morgan's LETM-like symptoms arguably first manifested within nine days of vaccination (although it is difficult to distinguish during this period between symptoms of his prior, non-neurologic problems and new distinct symptoms). Nevertheless, a timeframe of two to three weeks is both consistent with his causation theory as well as reasonable in light of other cases involving the onset of autoimmune demyelinating injuries, suggesting that the third *Althen* prong has been met. However, because the theory *itself*—that the flu vaccine *could cause* what the evidence established Petitioner was ultimately diagnosed with (NMOSD)—is evidentiarily deficient, the satisfaction of this single *Althen* prong does not assist Petitioner in satisfying his overall burden of proof.

[19] Although petitioners are not obligated to establish a mechanism in their efforts to prove entitlement, they often try to do so—and in such circumstances (as is the case here), it is reasonable to evaluate if they have succeeded. *See, e.g.*, *McKown v. Sec'y of Health & Human Servs.*, No. 15-1451V, 2019 WL 4072113, at *49 n.75 (Fed. Cl. Spec. Mstr. July 15, 2019).

literature indicating that NMOSD initially manifesting as LETM could be caused by a variety of infectious agents (i.e., the herpes virus, dengue fever, tuberculosis, etc.). Kim at 279–80. But this list does not also include the influenza wild virus. Nor did Petitioner's filings establish how an initial reaction to vaccination might be sufficient to create the kind of chronic, CNS-oriented inflammatory process that would ultimately morph into NMOSD. Thus, Petitioner has not offered sufficient reliable and persuasive evidence suggesting that the flu vaccine could cause a chronic form of CNS demyelinating disease such as NMOSD, that would unfold over a lengthy period of time.

Petitioner's efforts to satisfy the second *Althen* prong similarly founder on the facts—and especially my determination that his disease is best understood as NMOSD. Although the treaters who first saw Mr. Morgan post-vaccination may reasonably have understood his presentation to be comparable to LETM, over time it was evident that he more likely suffered from a related, but different, chronic condition. The record does not support the conclusion that the progression of Mr. Morgan's symptoms over a period of four or more years could reasonably be attributed to a vaccination received at the outset of that timeframe. Nor is it evident from the record that the vaccine, even if it had played some role in his initial presentation, continued to drive a pathologic process over such a lengthy period of time. Admittedly, several of Mr. Morgan's treating physicians reference the fact that he had received a flu vaccine one week prior to the onset of his symptoms. *See* Ex. 9 at 714, 897, 899; Ex. 10 at 1. But while these treaters may have so opined based on speculation that in turn deemed significant the temporal relationship between onset and vaccination, none later proffered the opinion that the vaccine could have caused the chronic condition of NMOSD. *See* Ex. 9 at 714, 897, 899; Ex 10 at 1. Again, consideration of the record's scope is not supportive of the conclusion that the vaccine caused Petitioner's overall course.

CONCLUSION

The Vaccine Act permits me to award compensation only if a Petitioner alleging a "non-Table Injury" can show by medical records or competent medical opinion that the injury was more likely than not vaccine-caused. Here, there is insufficient evidence to support an award of compensation, leaving me no choice but to hereby **DENY** this claim.

In the absence of a timely-filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accord with this decision.[20]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[20] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.